(4) Preventive *endocrine* treatment is a most fruitful medical field in incretory defectiveness, delinquency and presenility.

ARTERIAL DISEASES OF THE BRAIN AND CORD[1]

FOSTER KENNEDY

Professor of Neurology, Cornell University Medical College

All cells in the body can multiply and reproduce with the exception of those in the nervous system. These only are laid down in the embryonic life and growing as the individual becomes adult stretch out their processes in every organ and area of the body. When maturity of age has been attained, they no longer grow. They never reproduce themselves. Under these conditions, it follows that of all cells they are of necessity most long lived, but also of necessity they must alter with the changing condition of the entire organism. Some die long before their owner, and massive atrophy of the neural parenchyma is probably the most outstanding characteristic of the senile brain. However, one must emphasize that senility of structure is not produced by great age only; no individual over seventy-five is without alteration of brain tissue, but many persons twenty years younger have diffuse lesions in no way different from those of utter senility. Indeed, any severe infection or intoxication is capable of giving rise to cellular changes in the brain indistinguishable from those found in persons sorely stricken in years. Such infections act not only on the cells of the brain, but on the cells and fibres of nourishing blood vessels and by the devascularization thus produced secondary nerve changes are set up.

This change in blood vessels is altogether too causatively associated in our minds with the idea of age; much more should we think of it as due to all the infections and intoxications to which the body has been exposed throughout life. Such attacks have usually had but ingravescent and insid-

[1] Delivered October 3, 1928.

ious results and only after many years are these results made manifest in clinical symptoms and anatomical change. Therefore, in the majority of cases arteriosclerosis only appears in old age, but it is a product of many decades and we can all call to mind children and youths with arteries, as the saying is, of an old man. We are entirely too prone as a profession, to regard our current methods of research as ultimate and decisive and so to consider that no infection or intoxication exists in an individual if we have been unable to find it. By the same token psychiatrists too often assume a mental disease to be functional, "tooted in the mind," having a pathology in the lambent ether, a morbid process suspended somewhere, like Mahomet's coffin, between earth and heaven, for no better reason than that with our as yet crude methods, we have failed to find notable alteration in brain tissue. Let us remember for instance that only twenty years ago or less, Parkinson's disease was called functional, a neurosis, though we now know it is dependent on very precise changes in the cells of the lenticular nuclei. So far are we from having reached an ultima Thule in knowledge or technique that we must recognize that we have but lately embarked on our voyage of discovery.

As yet almost no progress has been made towards increasing the span of individual life. However, recognition of the fact that arterial degeneration is not the sure result of the inescapable years, but is a product of infections and intoxications occurring in those years, gives us a lively hope that Medicine may yet succeed in making life long by making life healthy. As man is conquering the foes in his environment, he may be trusted to conquer those within himself. In the meantime, however, arteriosclerosis is being manufactured in us poor ignorant ones, seldom in a generalized fashion, more often as localized lesions. Often advanced cerebral arterial change can be detected in persons lacking any sign of arteriosclerosis elsewhere, and the reverse situation also is found. Changes in the vessels of the brain give rise to a chain of focal lesions—areas of cerebral softening which are so often fatal. Such large

lesions are seldom primary. Long standing cerebral defects are always found whereby vessels are deteriorated and parenchyma made friable, atrophied, vacuolated; such alterations may not produce remarkable clinical symptoms, but they make almost inevitable a death by cerebral accident.

The senile brain is characterized by an amazing reduction in mass; the brain is retracted from the skull and from the membranes which are themselves often no thicker than tissue paper, and in which are frequently occurring osseous plaques. The meningeal vessels are spaced and small. The convolutions are thin and divided by deep sulci of greater size than the convolutions they surround. The convolutions are granular and wrinkled, and, most mysterious of all these conditions, the posterior third of the brain may be almost normal. We have no theory even on why the changes wrought by intoxications or old age should so persistently choose the frontal and parietal situations, but then we don't know why diphtheria should first strike part of the 3rd nucleus, why poliomyelitis should so avoid the posterior spinal horns, why encephalitis should be almost confined in lesions to the basal ganglia, or for that matter why tuberculosis should make a bilateral destruction of the adrenal bodies. Here again we need knowledge on infections and the forces which determine their paths of invasion.

The ventricles are always dilated in the aged, due less to increased cerebrospinal fluid than to the mass reduction already described which may reach 200 grams by weight. The corpus callosum as one would expect is slight, and is usually but a third its normal adult size—a reduction in association tracts which is not unrelated to the fading memory power of the aged. The cerebellum is affected though to a less degree than the frontal lobes; one case of reduction in cerebellar mass by two-thirds of its bulk has come under my observation, a man 82 with progressive bilateral cerebellar ataxia accompanied by intense general emaciation.

The nerve cell degeneration has been made especially clear by the use of Nissl's stains and the newer reactions of Ramon y Cajal and Hortega. The cells of the cortex are reduced in number showing that many have vanished utterly. Others, alongside healthy cells, are atrophic, pigmented or have undergone fatty degeneration, with increased production of neuroglial nuclei.

The large vessels are always atheromatous, especially those of the Circle of Willis. The smaller intracerebral vessels seldom lack lesions of different degrees of incidence, characterized by proliferation of the inner coat with shrunken lumen. Perivascular leucocytosis is the rule. Occasionally there is sclero-hyalin degeneration in which the entire wall is changed into a homogeneous hyalin mass, and again calcareous deposits and fatty degeneration may occur in the smaller capillaries.

Small focal lesions in cerebral tissue, some of microscopic size, are found as the result of vessel obliteration: as has been said these disseminations always precede the large focal lesions which produce paralysis and other easily recognized symptoms. Indeed, foci of miliary softening are often found scattered below the cortex, and these may be found side by side with perivascular gliosis. In the brains of persons who have suffered a senile dementia or even a number of transient cerebral palsies it is common to find lacunae with irregular walls containing granular debris or red blood cells.

It is uncertain whether senile dementia should be called a disease of the cerebral parenchyma or the cerebral vascular system. However, as our consideration of the morbid structure has just shown, it is almost impossible to divide the influences of the two associated deteriorations. The aged suffer a reduction in mental and physical power —there is an especial poverty in the faculty of creative imagining—many old people of genius or even talent have done mighty work—Titian, Michelangelo, and Voltaire come at once to the mind—but rarely do they create a new style, evolve a method different to that of their adult ma-

turity—old men of genius may soar magnificently on the Pegasus of their conditioned reflexes, but never do they find fresh horses! The person becoming senile has a loss in memory and especially for those memories most recently acquired. A loss of memory for nouns is pronounced and for proper names in particular, probably owing to the few associations united to such memories. He grows more and more egotistical and irritable. Somatic sensations especially from the gut play an increasing part in his mental and emotional life, and this increasing attention to his internal workings depresses his faculty of attention for his environment. Judgment is passed, therefore, on facts poorly received, suspicion is aroused and a sense of umbrage and persecution developed. The avarice of the aged is a return to the acquisitiveness of the young child, the whole mental life undergoes devolution so that nursery rhymes long forgotten may be the sole articulated product.

During this process in its beginning the senile is not without insight into his condition, he is forever on the defensive and talks jargon carefully to conceal his memory defects. His automatism sustains his front and sometimes cross examination exceeding the bounds of courtesy is needed to reveal the underlying poverty of ideas, attention or memory. Associated with these psychical alterations there is often vertigo, which may well be the result of true cerebral intermittent claudication, insomnia by night and sleepiness by day, transient apoplectiform attacks or short epileptic seizures with fleeting attacks of aphasia.

In the early stages, the patient is merely slowed up in his reaction time, enfeebled in his power to grasp abstract ideas and acutely aware of his condition. He fears insanity and complains of bagatelles. It should be stated here that certain cases of cerebral arteriosclerosis are indistinguishable by symptoms and almost so by autopsy from cases of general paralysis—the Wassermann reaction in the cerebrospinal fluid in such difficulties offers the only clue to correct diagnosis.

Apoplexy or severe cerebral thrombosis with retention of life and partial recovery and contracture are seen in the adult of late middle life—not in the aged. And for the simple reason that large lesions of destruction are usually fatal to the very old. That is not to say that hemiplegias of vascular origin cannot affect old people; they do, but the attack is less fulminant and the symptoms tend to regress. The hemiplegias of the old are less likely to be due to a blood effusion than to the breaking down of a lacuna in the brain. Pierre Marie warns us to be careful to avoid favorable prognoses in these lacunar hemiplegias; they depend for their production on diffusion of lesions in lacunar brains, which Marie describes as *vermoulé*—worm-eaten. The patient rarely falls unconscious in an attack, he gets dizzy and notices his hand and leg, or both, weaker and clumsier than usual, or he experiences notable difficulty in speech, a fleeting dysarthria. These symptoms improve and he may be well in a few days. In a week or two these symptoms may recur, perhaps on the opposite side of the body; the gait may be typically hemiplegic, but more probably it will be one of small quick steps (*Marche à petit pas*) in which each foot is advanced in front of its fellow by only a few inches. There may be retropulsion alternating with this odd little quick-step forward. The coordination of facial movements is impaired, the face may be asymmetrical, or one side of it may be incapable of emotional expression if the thalamus has been affected; a hemianopic quadrantic defect in the visual field may appear. Synergy and balance may be impaired, and in right sided hemiplegias there are dysarthrias and difficulty in swallowing. A peculiar and typical dysarthria is the necessity of forcible explosive repetition of key words in a sentence. "I want, want, want a knife, knife, knife." If the lacunar disintegration has been bilateral the picture produced may be that of pseudobulbar palsy with spasmodic involuntary laughing and crying—the emotional expression being usually irrelevant and inappropriate to the producing stimulus.

There is little to distinguish senile epilepsy from the

epilepsy of youth. Both major and minor forms occur, the aura and initial cry are less common, the coma after the fit more prolonged. Senile epilepsy, however, either produces or is accompanied by rapid mental enfeeblement. It is definitely a product of arteriosclerosis, and such brains are characterized by lacunar and senile plaques.

To have to listen to a narrative of the delapidation of the master organ of life is lugubrious and depressing; the picture looks like one of annihilation, a going down to oblivion and the dark shadows—but into that blackness man's spirit throws a mighty beam, for

* "If it be Life that waits we shall live forever unconquered.
If Death, then we shall die at last, strong in our pride and free."

MENOPAUSAL AND POST-MENOPAUSAL CONDITIONS IN WOMEN [1]

BENJAMIN P. WATSON

Professor of Obstetrics and Gynecology, Columbia University;
Director, Sloane Hospital for Women

The menopause is a very definite epoch in the life of a woman with which there is nothing comparable in the life of a man. Whilst it is determined by strictly physiological processes these may easily pass on to the pathological, and the physiological readjustments which take place may bring to light hitherto unrecognized and unnoticed dysfunctions in the different organs and systems in the body.

For an understanding of these pathological states it is necessary to have as clear an idea as possible as to what the menopause is, how it is brought about, and what are its normal effects on the various tissues, organs and systems of the body.

* Inscribed on Memorial Chapel to the Scottish War Dead, Edinburgh Castle.

[1] Delivered October 4, 1928.